mates of glomerular sieving coefficents used by Nordon et al [1] still rely on the amount of material measured post glomerular capillary wall. There is an urgent need to confirm the underlying assumption that this represents the actual quantity of material that has left the capillary and has been filtered.

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Red blood cells and thin basement membrane disease

To the Editor: The article by Collar et al entitled, "Red cell traverse through thin glomerular basement membranes" illustrates a red blood cell (RBC) passing through a small gap in the glomerular endothelium and basement membrane in a patient with thin basement membrane disease [1].

Phase contrast microscopy has demonstrated that up to 10,000 RBC/mL are normally present in the urine and that this number often increases to about 30,000 RBC/mL with vigorous exercise. The RBCs that occur normally and after exercise are dysmorphic in appearance, reflecting their glomerular origins [2, 3]. The basement membrane gaps described in thin basement membrane disease in Collar et al's report [1] may very well represent the normal means of RBC egress, and the increase associated with exercise could result from an increased number of membrane gaps or from the passage of more RBCs through each.

Although long suspected, the escape of RBCs through gaps in the glomerular membrane into Bowman's space has proved difficult to capture on electron microscopy. Most of our patients with thin basement membrane disease lose about 100,000 RBC/mL urine [4], which represents 2×10^8 RBCs a day. If a kidney has a million glomeruli, then on average 100 RBCs pass through the membrane of each glomerulus daily. Ultrastructural kidney sections comprise only a few glomerular loops, and Collar et al reflect that the membrane gaps are present for only 10 minutes [1]. Even with macroscopic hematuria, which corresponds to at least 5,000,000 RBC/mL urine [5], only 5000 RBCs pass through each glomerulus daily.

Can Collar et al also explain why many patients with thin basement membrane disease have hematuria but no proteinuria, which is different from the situation with most other glomerular lesions? Are the gaps in the membranes in thin basement membrane disease so small that negatively-charged plasma proteins are repelled from margins of the endothelial aspect of the similarly-charged membranes?

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ANCA-associated renal vasculitis

To the Editor: The Nephrology Forum discussed by Professor Savage reminded us of two items about ANCA-associated renal vasculitis (AARV): (*1*) the complex patho-